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## **Title page**

**Title:** Competition Intensity and Fatigue in Olympic Fencing

**Running head:** Physiology of fencing bouts

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# COMPETITION INTENSITY AND FATIGUE IN OLYMPIC FENCING

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## ABSTRACT

As yet, no studies have characterised fencing competitions. This was investigated in nine elite male foilists across two competitions, where countermovement jump (CMJ) height, testosterone (T), cortisol (C), alpha-amylase (AA) and immunoglobulin A (IgA), were obtained. Heart rate (HR) was measured throughout competitions and blood lactate (BL) and rating of perceived exertion (RPE) were measured post bouts. Average ( $\pm$  SD) scores for RPE, BL and HR (average, max and percentage of time  $\geq$  80% HRmax) were highest in the knockout bouts compared to poules ( $8.5 \pm 1.3$  vs.  $5.7 \pm 1.3$ ,  $3.6 \pm 1.0$  vs.  $3.1 \pm 1.4$  mmol/L,  $171 \pm 5$  vs.  $168 \pm 8$  bpm,  $195 \pm 7$  vs.  $192 \pm 7$  bpm, 74 vs. 68%) however, only significant ( $p < .05$ ) for RPE. CMJ height, albeit non-significantly ( $p > .05$ ), increased throughout competition and dropped thereafter. While responses of C, AA and IgA showed a tendency to increase during competition and drop thereafter (T and T:C doing the opposite), no significant differences were noted for any analyte. Results suggest that fencing is a high-intensity anaerobic sport, relying on alactic energy sources, however, some bouts evoke BL values of  $\geq 4$  mmol/L and thus derive energy from anaerobic glycolysis. High HR's appear possible on account of ample within and between-bout rest. The small competition load associated with fencing competitions may explain the non-significant findings found.

## INTRODUCTION

The sport of fencing has been investigated numerous times to describe the kinetics, kinematics and physical requisites of the attacking lunge (Guilhem, Giroux, Chollet, & Rabita, 2014; Turner, Chavda, Edwards, Brazier, Bishop, & Kilduff, 2016; Turner, et al., In press). As yet however, no studies have looked to describe competition intensity and residual fatigue. Such detail pertaining to biochemical and physiological changes can greatly inform training programme design and recovery strategy implementation. For example, measures of heart rate (HR), blood lactate (BL) and ratings of perceived exertion (RPE) taken within competition, can determine metabolic workload and the demands placed on energy metabolism (Haddad, Chaouachi, Castagna, Wong, Behm, & Chamari, 2011; Uchida, et al., 2014); RPE has recently been shown to be a valid method within fencing, showing high correlations ( $r = .73 - .99$ ) with HR-based methods (Bannister's and Edwards's TRIMP) across training sessions and competition bouts (Turner, Buttigeig, Noto, Marshall, Phillips, & Kilduff, In press). Saliva analysis can reveal the (physical and emotional) stress of competition (and requirements for rest and recovery) by describing hormonal fluctuations in testosterone (T) and cortisol (C) as previously reported in other sports (Cormack, Newton, McGuigan, & Cormie, 2008; McGuigan & Cormack, 2011; McLellan & Lovell, 2010), activation of the sympathetic nervous system through concentration changes in salivary alpha amylase (sAA) (Chatterton, Vogelsong, Lu, Ellman, & Hudgens, 1996; Chiodo, Tessitore, & Cortis, 2011), and signs of mucosal immunity depression through reductions in secretory immunoglobulin A (SIgA) (Neville, Gleeson, & Folland, 2008; Libicz, Mercier, Bigou, Le Gallais, & Castex, 2006).

For example, McLellan *et al.*, (2010) monitored the T:C response following a rugby league match and reported significant reductions which did not return to baseline values until 48 hours post match. This time point was suggested to be indicative of when training could safely resume without an increased risk of overtraining, injury or illness. Kivlighan and Granger (2006) showed that 2 km ergometer rowing increased sAA levels, and the magnitude of which was positively associated with performance. This trend has also been noted in marathon running (Ljungberg, Ericson, Ekblom, & Birkhed, 1997), triathlon (Steerenberg, van Asperen, Van Nieuw Amerongen, Biewenga, Mol, & Medema, 1997), 60 min cycle races (Walsh, Blannin, Clark, Cook, Robson, & Gleeson, 1999) and a taekwondo competition (Chiodo, Tessitore, & Cortis, 2011). Finally, the incidence of upper respiratory tract illness (URTI) is associated with increases in training load and a reduction in SIgA levels (Neville, Gleeson, & Folland, 2008; Libicz, Mercier, Bigou, Le Gallais, & Castex, 2006; Pyne, McDonald, & Gleeson, 2001); an association supported by longitudinal studies examining triathletes (Libicz, Mercier, Bigou, Le Gallais, & Castex, 2006), swimmers (Gleeson, McDonald, & Pyne, 2000) kayakers (Mackinnon, Ginn, & Seymour, 1993) distance runners (Mackinnon & Hooper, 1994), football players (Fahlman & Engels, 2005), and rowers (Neville, Gleeson, & Folland, 2008). Specifically, Neville *et al.*, (2008) reported that when SIgA concentration dropped below 40% of an athlete's mean healthy levels, they had a one in two chance of contracting an URTI within 3 weeks. Furthermore, 82% of the illnesses reported in a previous study were associated with a preceding decrease in SIgA (Fahlman & Engels, 2005). Given that illness ultimately results in the loss of training days or important competitions, coaches are understandably eager to use predictive measures.

Measures of stretch shortening cycle capability are considered indicative of neuromuscular fatigue (Johnston, Gabbett, Jenkins, & Julin, 2014; Johnson, Gibson, Twist, Gabbett, MacNay, & MacFarlane, 2013), with research showing that fatigue accumulation, normally lasting 48-72 hours post-exercise or competition, is detected through a continued deficit in jump performance (Cormack, Newton, & McGuigan, 2008; Johnson, Gibson, Twist, Gabbett, MacNay, & MacFarlane, 2013). For example, McLellan *et al.*, (2010) found that following a competitive rugby league, force-time data from a countermovement jump (CMJ) showed that peak rate of force development, peak power and peak force all dropped immediately after the match and lasted for 48 hours. These findings mimicked the body's stress response as measured by salivary C concentrations, and as such, salivary analysis coupled with measures of neuromuscular fatigue, may provide the temporal requirements to dissipate fatigue and return to full training without risking injury (Gabbett, 2004), illness (Neville, Gleeson, & Folland, 2008) and reductions in both competition and training performance (Elloumi, Makni,, Moalla, Bouaziz, Tabka, & Chamari, 2012).

Collectively therefore, all measures are proposed to combine to describe competition demand and the requirements for recovery, affecting exercise selection and the programming and periodisation of these. The aim of this study therefore, is to use all aforementioned measures to describe these demands within the sport of fencing, in order to inform training programme design. This is yet to be done and thus a necessity for evidence based practice. It is hypothesised that fencing will be deemed a largely anaerobic sport, inducing reductions in measures of power across the competition days, mirrored by the salivary analytes T, SIgA and SAA, with increases in C.

## **METHODS**

### **Experimental approach to the problem**

An observational research design was used as data was collected in actual competitions. Data was intended to inform future training and recovery strategies within elite fencing and thus results used to affect the wider fencing community. As such, scores for each athlete were averaged across two competitions to better enable the generalisation of results and controlling for the between-day fluctuations in variables. Data was collected across two competitions (an international and a national competition) spaced one week apart. Saliva samples and CMJ height were collected at the following time points: 48 h and 30 min pre-competition, 30 min after the poule stages, and 30 min, 48 and 72 h post competition. All data was collected between 0900 h and 0930 h with the exception of post poule and post knockout collection points, which were collected at ~ 1300 h and ~ 1900 h respectively. To avoid the acutely high scores consequent to the cortisol awakening response (Clow, Thorn, Evans, & Hucklebridge, 2004), fencers were requested to wake up at least one hour prior to saliva collection and to record time of day for both waking up and saliva collection, to check their compliance. Finally, on each competition day, fencers wore HR monitors throughout, and BL and RPE were taken following each bout. Fencers rested 24 h post-competitions, engaged in recovery sessions 48 hours post-competitions, and given the proximity of them, performed only light to moderate training sessions at all other time points, consisting of technical blade work, 5 point match sparring and reduced volume resistance training. Collectively these measures

describe competition demand and the requirements for recovery, affecting exercise selection and the programming and periodisation of these.

## **Subjects**

Nine elite male fencers (foil) gave written, informed consent to participate in this study. On average (mean  $\pm$  SD), fencers were  $22.3 \pm 2.8$  years of age,  $179.2 \pm 5.5$  cm tall,  $74.2 \pm 6.4$  kg in mass, and had  $14.3 \pm 3.6$  years fencing experience. All fencers were free from injury and of good fitness and health. Before the start of the study, all fencers attended a presentation outlining the purpose and benefits of the study and were familiarised with all test procedures. All procedures were granted ethical approval from Middlesex University, in accordance with the Declaration of Helsinki.

## **Procedures**

*Incidence and severity of illness symptoms:* Fencers were asked to complete a log book each morning, which asked for the following coded health problems: (1) no health problems today; (2) cold symptoms (runny stuffy nose, sore throat, coughing, sneezing, coloured discharge; (3) flu symptoms (fever, headache, general aches and pains, fatigue and weakness, chest discomfort, cough); (4) nausea, vomiting, and/or diarrhoea; (5) muscle, joint, or bone problems/injury; (6) other health problems (describe) (Nieman, et al., 2000; Nieman, et al., 2007). Severity was rated using the following scale: 1 = very mild; 2 = mild; 3 = moderate; 4 = strong; 5 = very



strong/severe. If fencers had cold or flu symptoms for a minimum of two consecutive days they were identified as symptomatic (Nieman, et al., 2007).

*Saliva Sampling Procedures:* Fencers were requested to collect 2 ml of unstimulated saliva via passive drool, into a cryovial for the analyses of C, T, SIgA, and sAA (Bishop & Gleeson, 2009; Proctor & Carpenter, 2007). To preserve the integrity of samples, fencers were requested to avoid food, fluid (except water) and brushing their teeth, one hour before collection; 10 minutes prior to collection, fencers had to rinse out their mouth with water (Groschl, Kohler, Topf, & Rauh, 2008). After collection, samples were immediately frozen at -20°C, before being transported to and stored at -80°C until analysis (Granger, Shirtcliff, Booth, Kivlighan, & Schwartz, 2004).

For SIgA and sAA, flow rates were calculated. Saliva collections were timed (s), to facilitate the calculation of saliva flow rate ( $Sal_{fr}$ ), as described elsewhere (Dimitriou, Sharp, & Doherty, 2002). Saliva density was assumed as 1.00g/ml (Walsh, Blannin, & A, 1999). SIgA and sAA flow rates were then calculated as the product of the absolute concentration of each and  $Sal_{fr}$  (mL/min) (Mackinnon & Hooper, 1994). Unlike the other tested biomarkers within this study, SIgA has been provided with a reference point to warn of a forthcoming risk of illness, to which sport scientist can take guidance (Neville, Gleeson, & Folland, 2008). Therefore the data of each athlete was also examined to identify drops below 40% of the baseline values (48 hours pre competition).

*Salivary Analysis:* All salivary analytes were analysed in duplicate via commercially available enzyme-linked immunosorbent assays (Salimetrics LLC, State College, PA,

USA) using an automated microplate reader (Fluostar Omega, BMG Labtech, Aylesbury UK). The assay ranges were: SIgA 2.5 - 600 µg/mL; sAA 3.28 - 980 iµ/mL; cortisol 0.33 - 83 nmol/L; testosterone 3.4 - 2080 pmol/L and sIL-6 2.08 - 100 pg/ml. The intra-assay CV was: sIgA 8.9%; sAA 5.2%; cortisol 5.3%; testosterone 5.3%; and sIL-6 1.3%. The inter-assay CV was: SIgA 11.2%; sAA 6.0%; cortisol 8.3%; testosterone 3.3%; and sIL-6 1.0%. Standard curves were constructed as per the manufacturer's instructions, and commercially available standards and quality control samples were used for the assays (Salimetrics LLC). All samples were analysed in the same series to avoid inter-assay variability.

*Neuromuscular Fatigue:* Neuromuscular fatigue and jump height was measured via a CMJ performed on a surface mounted force plate (type 92866AA, Kistler Instruments Ltd., Hook, United Kingdom). During CMJ trials, the athlete remained stationary on the plate for 3 s before jumping (enabling an accurate measurement of bodyweight). Vertical ground reaction force (VGRF) data was then averaged across this period and the jump was deemed to start when this value was reduced by 5 standard deviations (Owen, Watkins, Kilduff, Bevan, & Bennett, 2004). For analysis, the athlete's bodyweight was subtracted from the VGRF values. The vertical force impulse between the start of the jump and take-off was then calculated using the trapezoidal method (using intervals equal to the sample width) and in turn used to calculate take-off velocity (Owen, Watkins, Kilduff, Bevan, & Bennett, 2004). Jump height was finally calculated using  $v^2 = u^2 + 2as$ , where  $v$  = final velocity,  $u$  = initial velocity,  $a$  = acceleration and  $s$  = distance. All force data was filtered using a Butterworth fourth-order zero lag low-pass filter, with a cut-off frequency (20 Hz) determined by residual analysis (Winter, 2009).

*Heart Rate, Blood Lactate and Rating of Perceived Exertion:* Fencers wore HR monitors (Polar team<sup>2</sup> Pro, Warwick, United Kingdom) throughout the competition, where average HR, maximum HR (HRmax) and time spent above 80% HRmax was calculated. BL (mmol/L) (measured via finger prick of the non-fencing hand using a Lactate Pro) and RPE scores [using the Borg category ratio 10-point scale (Borg, 1982)] were taken 5 min after each bout; the former was also taken prior to the start of the competition and all scores were averaged across both competitions, and separated to define poule bouts (first to 5 hits) and elimination bouts (first to 15 hits). Furthermore, scores were also analysed to determine if increases were noted following each bout, as the competition progressed.

### **Statistical Analysis**

Measures of normality were assessed using the Shapiro-Wilk statistic. To determine the reliability of jumps, single measures intraclass correlations (two-way random with absolute agreement) and the coefficient of variation were calculated. Repeated measures ANOVA with bonferroni correction were performed to investigate temporal changes in CMJ and biomarker values; this test is also considered valid for non-parametric data (Field, 2013). During pilot testing large inter-individual variations were noted in salivary analyte concentrations and thus it was anticipated that these would ultimately invalidate significance testing. Therefore, effect size analysis was also used (Hopkins, 2004) and interpreted according to Rhea (2004), with athletes classed as “highly trained”. Differences in RPE, HR and BL values, between poules and knockouts, were assessed using a paired samples *t*-test. Pearson’s Product Moment Correlation was used to investigate associations between biomarkers and

CMJ data. All statistical analysis was conducted using SPSS version 21 with the level of significance set as  $p < 0.05$ .

## RESULTS

All data was reliably assessed, with CMJ height producing an ICC (95% CI) of 0.95 (0.92-0.96) and CV values of 2.9%. Only C was not normally distributed and thus when assessed for relationships with all other variables, data was ranked and analysed using Spearman's Correlation Coefficient.

No significant correlations amongst variables were noted. Scores for RPE, BL and HR are presented in Table 1, where values for each are highest in the knockout rounds compared to the pools, however, only is this difference significant in time of bout and RPE. Scores for each variable did not show a trend of increasing subsequent to each bout.

**Table 1.** About here

CMJ height increased throughout the competition and dropped thereafter, changes however, were not significant. Relative to 48 h pre-competition scores, ES interpreted changes were classed as “moderate” through to 48 h post-competition, and then a “small” change at 72 h post-competition (Fig 1a). While C, T and sAA appeared to increase during competition and drop thereafter, SIgA and T:C showed the opposite; no significant differences were noted across time-points for any biomarker (Figure 1b

– f). ES analysis however, did reveal “large” changes in C and T:C, “moderate” changes in SIgA and sAA and “small” changes in T. ES values and descriptors are illustrated in Figure 1.

On average, SIgA flow rate (SIgA<sub>fr</sub>) never dropped below 40% of baseline values (48 h pre-competition; identified via the dashed line in Figure 1f). However, on an individual basis, 6 of the 9 fencers did on at least one occasion, with 2 athletes remaining below this threshold during and post competition. These two and one other fencer of the 6, also reported a URTS on at least two consecutive days and were thus classed as symptomatic (Nieman, et al., 2007).

**Figure 1a-f.** About here

## **DISCUSSION**

This is the first study to monitor the physiological intensity of a fencing competition and the time course restoration of its inherent fatigue; this information can inform the training programme design of these athletes and their requirements for recovery post competition. Our hypothesis was only partially correct. While we conclude that fencing is an anaerobic, and largely alactic sport, we found no evidence of significant physiological fatigue; these findings are likely to challenge the traditional approaches to fitness training for these athletes. Scores for RPE, BL and HR (max and > 80% max) were highest in the knockouts compared to the poules (see Table 1), with differences in perceptions of RPE being significantly different between the two. CMJ

height increased throughout the competition including immediately after, before declining below baseline thereafter; changes were not significantly different, however ES analysis did reveal these changes as moderate. Changes in biomarker concentrations were also not significantly different throughout the testing period, although C, T and sAA<sub>fr</sub> appeared to increase during competition and drop thereafter, with SIgA<sub>fr</sub> and T:C showing the opposite. ES analysis revealed that these changes were meaningful.

### **Competition intensity**

The high and sustained HR values, coupled with high RPE scores suggest that fencing (foil) is a high-intensity anaerobic sport, and for the most part, relies on alactic energy sources (i.e., phosphocreatine). That said, the spread of data (i.e., the SD) suggests that some bouts (both poules and KO's) evoke BL values of  $\geq 4$  mmol/L and thus derive energy from anaerobic glycolysis.

A large percentage of poule and KO bouts are spent above  $> 80\%$  HR<sub>max</sub> (68 and 74% respectively), which is surprising given the length of each (5.33 and 15.09 min respectively). However, given the ample opportunity for rest within foil fencing, with work to rest ratios reported as 1 : 3 (5 s work to 15 s rest) (Roi & Bianchedi, 2008), this may not be a surprising finding and may also explain how BL values, on average, remained  $< 4$  mmol/L. Although only an anecdotal observation, fencers can also prolong within-bout rest periods through methods such as “fixing” the equipment responsible for electronic scoring, realigning swords, and tampering with protective clothing for example. It should also be noted that while a fencing competition lasts around 10 h (Roi & Bianchedi, 2008), actual bout time only accounts for  $\sim 10\%$  of

this, and there can be anywhere between 15 and 180 min between bouts (Roi & Bianchedi, 2008). Therefore there is also sufficient opportunity to rest and recover between bouts, which one would assume if done correctly, would provide adequate time (given the brevity of bouts) to alleviate much of the residual fatigue. Finally, scores for RPE, HR and BL did not appear to increase following each bout; if an accumulation of fatigue was present, this may be an expected observation. It is more likely that the opponent dictates each bout's intensity. Bouts that are won or lost easily would be less intense than those that are evenly matched and thus last longer, also possibly evoking psychological stress and anxiety around the uncertainty of the result. Subsequent to this, there may be increased hypothalamic-pituitary-adrenal axis activity (HPA) and sympathetic nervous system (SNS) activity (Ader, Cohen, & Felten, 1995), which in turn could lead to increased cortisol and AA output, cardiovascular response, fatigue and increased risk and susceptibility to infection (Pedersen, Zachariae, & Bovbjerg, 2010), again indices not noted here. This finding is perhaps unsurprising during the poule stages as the competition is less evenly matched, i.e., it is possible to go from a close match (e.g., 5 – 4 on points) to an easy match (e.g., 5 – 0) and thus experience greater relative recovery. It is a more surprising finding during the knock-out stages, when only the better competitors are left and matches are more evenly contested. As aforementioned however, it may simply be that there are enough breaks between bouts to not carry over residual fatigue regardless of opponent or stage of competition. Also, the tested participants were elite and fitness training involves conditioning work that is designed to exceed that of competition; results here may support the efficacy of this training.

## **Neuromuscular fatigue**

Due to the expected muscle damage and soreness associated with fencing, assumed on the basis of performing a high frequency (140 per competition) of lunges (Roi & Bianchedi, 2008), with associated high landing forces ( $> 3$  times body weight) and eccentric muscle force (Guilhem, Giroux, Chollet, & Rabita, 2014; Turner, Chavda, Edwards, Brazier, Bishop, & Kilduff, 2016), CMJ scores were expected to drop throughout the competition and remain below baseline for as long as 72 h after (McLellan & Lovell, 2010). In fact, CMJ height actually increased during competition (and moderately so according to ES analysis) and immediately after. Therefore results here actually found a potentiating effect of competition, presumably linked to muscle temperature (Wright, Hull, & Czeisler, 2002) and the psychological arousal and concomitant excitability of the nervous system (French, et al., 2007; Viru, Viru, & Bosco, 2003); both of which seemingly outweighed fatigue. The increases in CMJ height are supported by the increases seen in both cortisol and sAA<sub>fr</sub>, markers of SNS excitability (Chatterton, Vogelsong, Lu, Ellman, & Hudgens, 1996; Chiodo, Tessitore, & Cortis, 2011). Assuming that muscle damage is present, then CMJ height may not be indicative of this, or there was limited damage on account of the repeat bout effect response (McHugh, 2003; Clarkson, Nosaka, & Braun, 1992) and further indication of the highly trained status of the tested athletes. It should also be noted however, that while the CMJ is generally regarded as useful fatigue-monitoring test, Gathercole et al., (2015) recently showed that the same fatiguing stimuli can elicit markedly different effects between individuals and across CMJ variables e.g., peak power, impulse, eccentric and concentric duration and flight time. Therefore neuromuscular fatigue may also manifest itself as an altered movement strategy rather than just a reduction in CMJ output, and as such, the use of a full CMJ variable



battery appears most prudent for sensitive NM-fatigue detection (Gathercole, Sporer, Stellingwerff, & Sleivert, 2015). Future studies should therefore investigate fatigue with respect to these variables.

It would appear that competitions do not involve acutely significant central nervous system or peripheral muscle fatigue in elite fencers; a statement supported by HR, BL RPE and CMJ data. Fatigue appears to have been masked by central nervous system excitability, potentiation that may be associated with muscle temperature increases and between bout recovery and nutritional interventions associated with elite athletes. Interestingly however, CMJ scores showed a small decline (via ES analysis) following the competition that was still apparent 72 h post-competition. Also, similar observations were made regarding SIgA<sub>fr</sub> (discussed later), where 3 of 9 fencers could also be diagnosed with a URTI through logbook reviews of health problems. As such, some caution must be exerted during training at this point.

### **Salivary analysis**

Cortisol and Testosterone are considered valid markers of training load (Cormack, Newton, & McGuigan, 2008; McLellan & Lovell, 2010), with the latter described as the primary anabolic marker for protein signaling and muscle glycogen synthesis, and the former a stress hormone which suppresses immunity, mediates catabolic activity, increasing protein degradation and decreasing protein synthesis in muscle cells (Cormack, Newton, McGuigan, & Cormie, 2008). Cortisol is also associated with anxiety (Ader, Cohen, & Felten, 1995), HPA activity (Dimitriou, Sharp, & Doherty, 2002), depression and creatine kinase, which is a marker of muscle damage (Kraemer, et al., 1993). The non-significant increases in C levels noted herein are in contrast to that reported in rugby league (McLellan & Lovell, 2010), rugby union (Elloumi, Maso,

Michaux, Robert, & Lac, 2003), soccer (Kraemer, et al., 2004), American football (Hoffman, et al., 2002) and swimming (Dimitriou, Sharp, & Doherty, 2002) for example. However, it is clear that values did increase, especially when considering that the within competition measurements would typically be lower than early morning measurements on account of circadian variation (Reilly, Atkinson, & Waterhouse, 1997); these assertions are supported by the large changes noted during competition as revealed by ES analysis (Fig 1b). The individual variation and high variability of scores between athletes, seen here and elsewhere (Dimitriou, Sharp, & Doherty, 2002), also left it unlikely that statistically significant differences would be noted. Furthermore, given our findings regarding actual exercise duration, results are in support of Hill *et al.*, (2008) who found that while increases in C are dependent on exercise intensity ( $\geq 60\%$  of maximal oxygen uptake), secretion is also dependant on exercise duration, at least 20–30 min is required, and the biofeedback regulation by the HPA axis (Tharp & Barnes, 1990; Törnhaage, 2009). While above this relative threshold large elevations in blood C levels can occur, insignificant changes are noted below this. Furthermore, the 30 min pre-competition C levels appeared higher than the 48 h pre-competition, suggesting anticipatory stress to competition (Kraemer, et al., 1993; Ader, Cohen, & Felten, 1995). Based on the biofeedback regulation theory, the 30 min pre-competition C levels perhaps were already too high for exercise to induce a significant response (Dimitriou, Sharp, & Doherty, 2002). However, increases in C have also been found in a kickboxing (Moreira, Arsati, & Lima-Arsati, 2010) and wrestling (Coelho, Keller, & da Silva, 2010) match. While this may be on account of muscle damage, the rise in C has also been suggested to coincide with the onset of blood lactate accumulation (Ratamess, et al., 2005; Port, 1991) and our findings reveal that on average, they operate under this threshold. Collectively these

findings also support the (non-significant) changes found in T, which shares similar volume load thresholds to C (Linnamo, Pakarinen, Komi, Kraemer, & Häkkinen, 2005; Lu, et al., 1997). Furthermore, T release has been found to correlate to a high strength training age (i.e.,  $\geq 2$  years strength training experience) (Kraemer, et al., 1992) and strength capacity (e.g., being able to back squat  $\geq 2$  times body weight) (Crewther, Cook, Gaviglio, Kilduff, & Drawer, 2012), factors that the tested athletes did not meet. Given these findings, T:C providing an indication of the anabolic/catabolic balance in response to training and competition (Cormack, Newton, & McGuigan, 2008), also provided no significant changes. In fact, T:C increased post-competition, largely on account of the drop in C; this drop may be attributed to the reduction in competition anxiety (Kraemer, et al., 1993). Again, given that T and C exhibit diurnal variations whereby concentrations are typically higher in the morning and drop throughout the day (Lejune-Lenain, Van Cauter, Desir, Beyloss, & Franckson, 1987), it may be that there was some elevation in recorded levels, but these were offset by the natural decline in release patterns occurring late in the afternoon and evening, when samples were taken at competition. Finally, post-competition values suggest that athletes can begin full training again 72 hours post-competition.

Salivary alpha amylase monitoring, like C, reflects the stress response to psychological and physical stress (Nater, et al., 2006; Kivlighan & Granger, 2006; Granger, et al., 2006). However, unlike C which represent the slower endocrine response to stress (i.e., release via the HPA axis), sAA represents the faster activation of the sympathetic branch of the autonomic nervous system (ANS) and the release of catecholamines (Chrousos & Gold, 1992); collectively therefore, they may provide a

more precise prescription of training and recovery cycles in athletes (Papacosta & Nassis, 2011). Unlike C, which is transported from blood to saliva, sAA is produced locally in the salivary glands and controlled by the ANS (Chatterton, Vogelsong, Lu, Ellman, & Hudgens, 1996; Skosnik, Chatterton, Swisher, & Park, 2000), and given that physical exercise causes activation of the SNS, it is expected that sAA will display increases in response to exercise (Kivlighan & Granger, 2006). Such observations have been reported previously as aforementioned. Again, increases in sAA appear mostly dependent on exercise intensity (Bishop & Gleeson, 2009) with a relationship between measures of sAA and blood lactate also reported (de Oliveira, Bessa, & Lamounier, 2010); perhaps these findings support why we did not note significant changes. That said, changes were regarded as moderate, but again we should note that sAA exhibits a pronounced decrease within 60 min after awakening and a steady increase of activity during the day (Nater & Rohleder, 2009). We must therefore acknowledge that the increase seen in the sAA<sub>fr</sub> values might have also been attributed to circadian variations.

SIgA functions as the first line of defence to viral pathogens entering the body via mucosal surfaces (Mazanec, Nedrud, & Kaetzel, 1993), thus acting to prevent infections of the upper respiratory tract. Nieman (1994) reported a “J-shaped” relationship between training load and susceptibility to URTI’s, where decreases in SIgA accompany a high training load (Neville, Gleeson, & Folland, 2008; Libicz, Mercier, Bigou, Le Gallais, & Castex, 2006) thus increasing its incidence; low levels of physical activity also increase risk whereas moderate levels provide a protective effect. Short bouts (< 30 min) of high intensity exercise (> 80% VO<sub>2</sub>max) have also been found to increase SIgA concentration (Bishop & Gleeson, 2009; Nieman, 1994) and typically, assuming testing does not follow strenuous long-term training, SIgA

recovers within 24 h post-exercise (Bishop & Gleeson, 2009). Here and albeit non-significant, SIgA<sub>fr</sub> showed moderate decreases during the competition, which had not returned to baseline 72 hours later. Also, considering SIgA<sub>fr</sub> is subject to a morning nadir in circadian release patterns, with levels rising throughout the day (Dimitriou, Sharp, & Doherty, 2002) and coupled with large increases in the immunosuppressive hormone C, findings appear more meaningful. That said it may not be until SIgA levels drop below 40% of baseline values that athletes are at greater risk of illness and infection and a so called “open window” is thus exposed (Neville, Gleeson, & Folland, 2008). On average, SIgA<sub>fr</sub> never dropped below 40% of baseline values. However, on an individual basis, 6 of the 9 athletes did on at least one occasion, with 2 athletes remaining below this threshold, and three reporting URTS on at least two consecutive days. Furthermore, our findings showed that 50% of those fencers with SIgA<sub>fr</sub> below 40% of baseline values were also URTI symptomatic at least during the post-competitive period, supporting previous studies that reported that ~ 95% of all infections start at mucosal surfaces (Engeland, Hugo, Hilgert, Nascimento, Junges, & Bosch, 2016). Our data appears to support the notion of a one in two chance of contracting an URTI when mean healthy levels of SIgA drop below 40% (Neville, Gleeson, & Folland, 2008). Nevertheless, biomarkers associated with illness development, either previously reported or within the present study, do not guarantee whether a person will stay healthy or develop illness, which further supports the multifactorial nature of immunity.

## **PRACTICAL APPLICATIONS**

The between bout timings of a fencing competition are unpredictable as is the quality

of opposition, thus it is advisable to prepare athletes for the worst-case scenario; a short break followed by a maximum point bout (i.e., 29 hits) on account of an evenly contested match. In this scenario, RPE is likely to be  $> 8$  and BL  $> 4$  mmol/L, and given the nature of the fight, high-intensity interval training is recommended in preparation for this, ensuring that athletes are exposed to high concentrations of BL, building a buffering capacity and tolerance of hydrogen ions as a consequence.

Our results appear to show that the tested fencing competitions did not evoke significant acute central or metabolic fatigue in elite fencers. The lack of within competition fatigue may not be surprising given the format of a fencing competition, which provides ample opportunity for recovery. In fact, and assuming consistent psychological stress and appropriate nutrition, it should be conceivable that fencers can fence at maximal intensity throughout the duration of the competition. Subsequently, and beyond strength and conditioning practices, the sport science support teams of these athletes should investigate various recovery strategies around fuel and fluid replacement (i.e., nutrition) and psychological interventions to cope with the high stress that may in turn increase intensity and fatigue.

Finally, given the small risk (albeit still above 40% of baseline values) indicated by reduced SIgA<sub>fr</sub>, recovery based sessions and training up to and including 72 h post-competition, could consider commencing training from late morning or early afternoon. This is due to circadian rhythms whereby C (an immunosuppressive) is highest in the morning and SIgA, which is already below baseline, is at its lowest. The high inter-individual and intra-individual variability of the selected biomarkers' response to exercise seen in this study reinforces the importance of individual monitoring especially of elite athletes.

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**Figure 1a-f.** While countermovement jump height (Fig 1a), cortisol (Fig 1b), testosterone (Fig 1c) and salivary alpha amylase (Fig 1e) appeared to increase during competition and drop thereafter, testosterone to cortisol ratio (Fig 1d) and  $S_{fr}$  (Fig 1f) showed the opposite, and no significant differences were noted across time-points for any of the measured salivary biomarkers. Magnitude of change is identified using effect size (ES) analysis and interpreted according to Rhea (2004), where T = trivial, S = small, M = moderate and L = Large. ES scores represent changes from 48 hours pre-competition. Error bars represent the standard deviation. The dashed line in Fig 1f represents the value corresponding to 40% of baseline. According to Neville et al., (2008) when values drop below this, they have a one in two chance of contracting an upper respiratory tract infection.

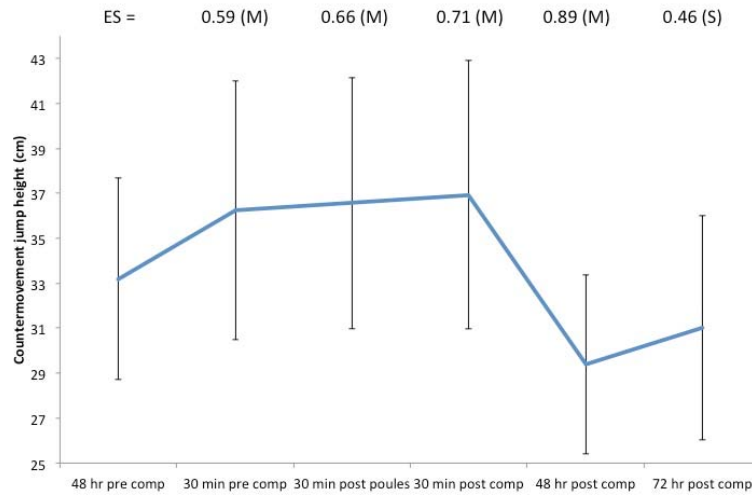


Figure 1a

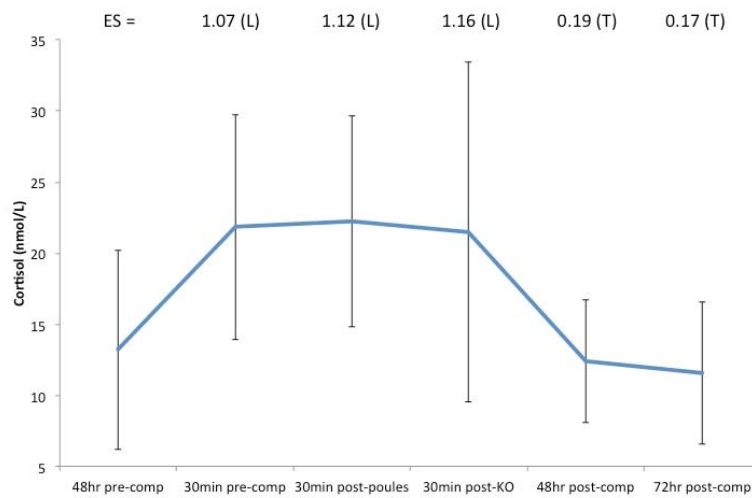


Figure 1b

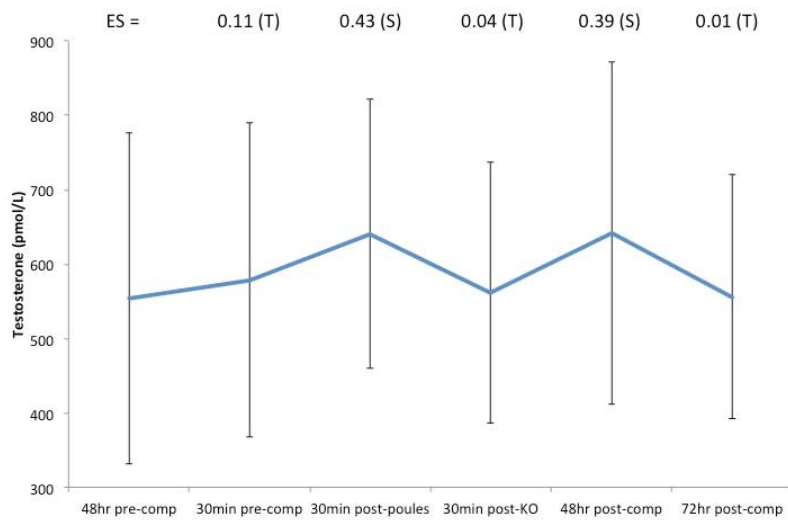


Figure 1c

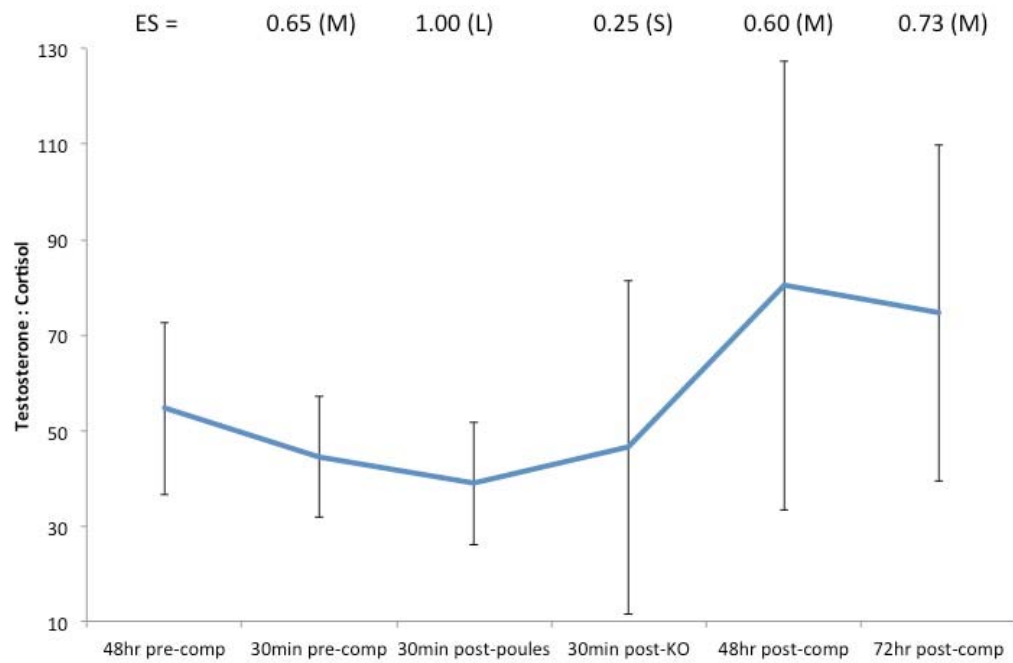


Figure 1d

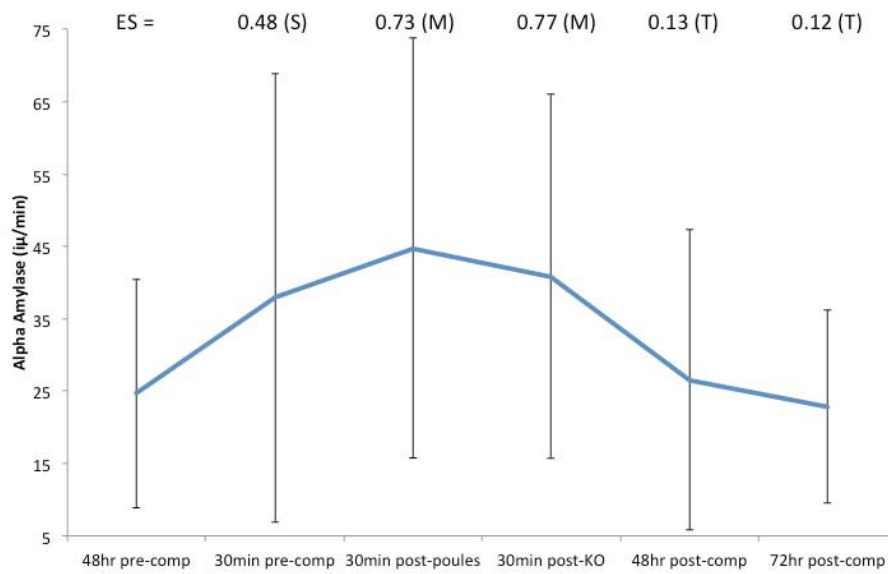


Figure 1e

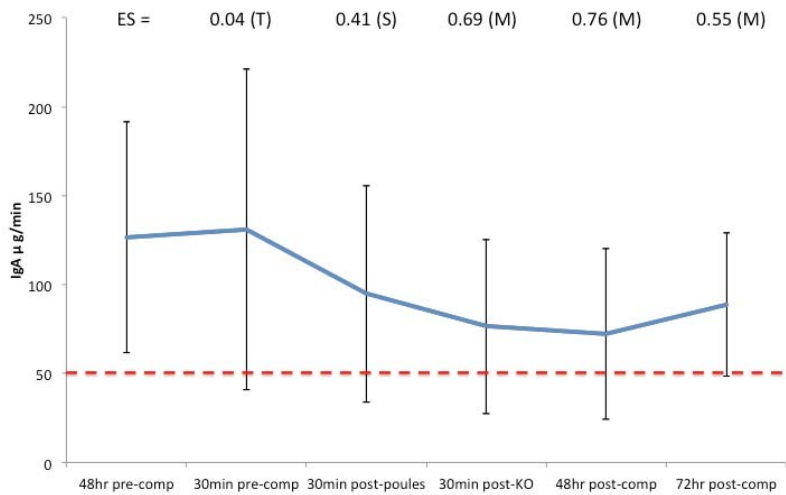


Figure 1f

**Table 1 Mean ( $\pm$ SD) results from two competitions, separated according to pool and knockout stages**

	Time (min)	RPE	BL (mmol/L)	HRave (bpm)	HRmax (bpm)	>80%HRmax
Pools	5.33 $\pm$ 2.15	5.7 $\pm$ 1.3	3.1 $\pm$ 1.4	168 $\pm$ 8	192 $\pm$ 7	68%
Knockout	15.09 $\pm$ 5.24*	8.5 $\pm$ 1.3*	3.6 $\pm$ 1.0	171 $\pm$ 5	195 $\pm$ 7	74%

**Key:** Time = length of bout in minutes; RPE = rating of perceived exertion; BL = Blood lactate;

HRave = average heart rate (HR); HRmax = maximum HR; >80%HRmax = percentage of time

spent above 80% of HRmax. \* = Significantly different from pool bouts.